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# Public policy, institutional cognition, and the geographic diffusion of multiple-drug-resistant HIV in the United States

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## Abstract

Public policy and economic practice, both quintessential expressions of institutional cognition, create an opportunity structure constituting a tunable, highly patterned, 'non-white noise' in a generalized epidemiological stochastic resonance which can efficiently amplify unhealthy conditions within marginalized populations to evoke infectious disease outbreaks. This is particularly true for infections carried by socially-generated 'risk behaviors'. A number of local epidemics originating in such keystone communities may subsequently undergo a structure-driven phase transition to become a coherent pandemic, a spreading plague which can entrain more affluent populations into the disease ecology of marginalization. Here we apply this perspective, which is formally homologous to recent theoretical developments in cognitive psychology, to the forthcoming social and geographic diffusion of multiple drug resistant (MDR) HIV from current AIDS epicenters to the rest of the United States.

HIV is an evolution machine, easily able to mutate around the selection pressures generated by both microbicides and vaccines. Effective control of the pathogen will require a return to traditional public health strategies aimed squarely at improving living and working conditions. Such interventions provide a strategic, ecosystem-wide, detuning of the synergistic factors generating a coherent, socially and geographically diffusing, behaviorally-transmitted infection. Broad, traditionally progressive, public policy is needed to significantly improve the embedding opportunity structure and spatiotemporal stability of communities in which AIDS is a primary index of marginalization.

MDR-HIV is poised to spread across the US in much the same manner as the early, pre-HAART, stages of the pandemic. Absent significant regime change, the Katrina disaster affecting New Orleans provides a sentinel case history for the likely outcome.

**KEY WORDS** Apartheid, information theory, phase transition, renormalization, recurrent epidemic, social inequality, stochastic resonance.

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**1. INTRODUCTION** Affluent subpopulations in the US have benefited greatly from the introduction of highly active antiretroviral therapy (HAART) against HIV: from 1995 to 1997, for example, HIV/AIDS deaths declined 63 % in New York City, primarily among middle-class, and highly organized, Gay males (Chiasson et al., 1999). Declines in AIDS deaths have otherwise been quite heterogeneous, depending critically on both the economic resources and community stability of affected populations (e.g. R.G. Wallace, 2003). At present, AIDS deaths in the US are, largely, another marker of longstanding patterns of racism and socioeconomic inequity (e.g. Wallace and McCarthy, 2006). Those who have economic resources, or reside in stable communities not subject to various forms of 'redlining', have effective access to HAART, others do not.

HIV is, however, an evolution machine (e.g. Rambaut et al., 2004) which, at the individual level, almost always develops multiple drug resistance, resulting in overt AIDS and subsequent premature fatality. Such response to chemical pesticides, as has been the case with myriad other biological pests, is now becoming manifest at the population level. By 2001 in the US some 50 % of patients receiving antiretroviral therapy were infected with viruses that express resistance to at least one of the available retroviral drugs, and transmission of drug-resistant strains is a growing concern (Clavel and Hance, 2004; Grant et al., 2002). MDR-HIV is, in fact, rapidly becoming the norm, and the virus may even develop a far more virulent life history strategy in response to the evolutionary challenges presented by HAART, its successor microbicide strategies, or planned vaccines (R.G. Wallace, 2004), a circumstance which may have already been observed (e.g. Simon et al., 2003).

The review by Rambaut et al. (2004) puts the matter thus:

> "HIV shows stronger positive selection than any other organism studied so far... [its viral] recombination rate... is one of the highest of all organisms... Within individual hosts, recombination interacts with selection and drift to produce complex population dynamics, and perhaps provide an efficient mechanism for the virus to escape from the accumulation of deleterious mutations or to jump between adaptive peaks. Specifically, recombination

might accelerate progression to AIDS and provide and effective mechanism (coupled with mutation) to evade drug therapy, vaccine treatment or immune pressure... More worryingly, there is evidence that some drug-resistant mutants show a greater infectivity, and in some cases a higher replication rate, compared with viruses without drug resistant mutations."

MDR-HIV is already emerging in the very epicenters and epicenter populations where HIV itself first appeared (Clavel and Hance, 2004), since these were the first to benefit from HAART, and thus seems likely to follow diffusion patterns similar to those of the earlier stage of the AIDS epidemic. We begin with a reconsideration of that period.

**2. INITIAL GEOGRAPHY OF AIDS** Infectious disease is often seen as largely a marker for underlying urban structure. For example, Gould and Tornqvist (1971, p. 160) write:

> "As the urban lattice hardens, and the links between the major centers strengthen, the dominant process is apt to change from a [spatially] contagious to a hierarchical one.
>
> We have few examples of this dramatic change in innovation diffusion, but one particularly striking one comes from the early history of the United States (Pyle, 1969). The disease cholera is hardly an innovation we would like to spread around, but it does form a useful geographical tracer in a spatial system, rather like a radioactive isotope for many systems studied by the biological sciences. The first great epidemic struck in 1832 at New York and Montreal, and then diffused slowly along the river systems of the Ohio and Great Lakes. A graphical plot of the time the disease was first reported against distance shows a clear distance effect, indicating that basically processes of spatial contagion were operating. A plot of time against city size shows no relationship whatsoever. However, by 1849, the rudimentary urban hierarchy of the United States was just beginning to emerge. The second epidemic struck at New York and New Orleans in the south, and a plot of first reporting times against city size, indicates that a hierarchical effect was beginning to structure innovation flows at this time. Finally, in 1865, when the third epidemic struck, the railways were already strengthening the structure of America's urban space. The disease jumped rapidly down the urban hierarchy, and a plot of reporting time against city size shows that a very clear hierarchical process was at work."

The first stages of the AIDS pandemic in the US seem to provide a modern example. The cover of Gould's 1993 book *The Slow Plague*, with more detail in Gould (1999), presents a time sequence of maps showing the number of AIDS cases in the US on a logarithmic scale. Cases first appear in the largest US port cities: New York, Los Angeles, San Francisco, Miami and Washington DC. Subsequent spread is by hierarchical hopscotch to smaller urban centers, followed by a spatially contagious winestain-on-a-tablecloth diffusion from city center into the surrounding suburban counties.

Figure 1, from Wallace et al. (1999), gives a detailed analytic treatment of the hierarchical hopscotch. Using multivariate analysis of covariance, it shows the log of the number of AIDS cases in each of the 25 largest US metropolitan regions for two periods, (1) through April, 1991 and (2) from April, 1991 through June, 1995, as functions of a composite index defined in terms of a region's local pattern of susceptibility and its position in the US urban hierarchy. The local indices are (i) the log of the number of violent crimes in the region for 1991, and (ii) an index of 'rust belt' deindustrialization, the log of the ratio of manufacturing employment in 1987 to that in 1972. The global index, of position on the US urban hierarchy, is the log of the probability of contact with the New York City metro region, the nation's largest, determined from a county-by-county analysis of migration carried out by the US Census for the period 1985-1990.

Locally, high levels of violence and industrial displacement represent bust-town and boom-town social dynamics leading to the loosening of social control. Nationally, the probability of contact with New York represents inverse socio-spatial distance from the principal epicenter of the US AIDS epidemic. Multivariate analysis of covariance finds the lines for the two time periods are parallel and each accounts for over 90 percent of the variance in the dependent variate. Thus later time periods are obtained from the earlier simply by raising the graph in parallel. We take this as representing a propagating, spatio-temporally coherent epidemic process which has linked disparate, marginalized 'core groups' inner-city neighborhoods of Gay males, intravenous drug users, and minority youth across time and space with the rest of the urbanized US, ultimately placing some 3/4 of the nation's population at increasing risk. See Wallace, Wallace et. al (1999) for details.

Analysis of the spread of AIDS in the 24-county New York Metropolitan region – the dominant node in the US urban hierarchy – based on the pattern of commuting into Manhattan, shows an analogous coherence. Figure 2, taken from Wallace et al. (1995), shows the log of AIDS cases per 100,000 population for each of the counties for three periods (1), through 1984, (2) 1985-87, and (3) 1988-1990, as a function of a composite index made up of (i) a local susceptibility factor, the percent of a county's population living in poverty in 1980, and (ii) the area density of the equilibrium distribution of a Markov process constructed from the 1980 county-to-county commuting pattern, as determined from US Census data.

Again, multivariate analysis of covariance finds the three time periods parallel, but displaced vertically, as the disease changed from one of primarily Gay middle-class males into one equally distributed between Gay males and poor, largely minority intravenous drug users.

Examination of several other metropolitan regions (Wallace et al. 1997) shows similar coherence in city to suburban spread of disease for the hollowed out urban areas of the US,

i.e. those in which policies of planned shrinkage or benign neglect resulted in great devastation of minority voting blocks, for example Detroit, Washington DC, and Philadelphia.

Remarkably, however, the San Francisco metropolitan region, which contains one of the principal US AIDS epicenters – the Gay ghetto of the Castro District in San Francisco itself – displayed a far different pattern, in which each of the region's individual counties seems to have its own independent epidemic, obviously seeded from the center, but with no apparent regional coherence. This difference merits some further discussion, and its explanation is one of the foci of this work.

Wallace et al. (1997) defined the AIDS outbreak near a central city as 'regionalized' in terms of a Markov process inferred from the commuting pattern linking the central city and its suburban counties, and the counties with each other. If a metro region had $n$ counties, an $n \times n$ matrix was constructed from Census data of the fraction of the workforce commuting from each county into each other county, including itself. Since the row sums were automatically normalized to unity, an associated Markov process could be defined, and the long-time equilibrium distribution calculated.

The essential independent empirical parameter was the equilibrium distribution per unit area for each of the $n$ counties of the metro region.

The AIDS outbreak was characterized as regionalized if a regression of the log of the AIDS cases per unit population against this independent variate – log of the equilibrium distribution per unit area – was significant in the absence of the commuting center, Manhattan (or New York County) and San Francisco county, respectively. Unlike the analysis of figure 2, local indices were not added to the regression model.

The AIDS pattern for the New York Metro Region was highly significant, in this sense, with an associated $R^2$, the fraction of variance accounted for by the regression model, of 76.3 % through 1990, while that for San Francisco was, remarkably, not significant at all, having an $R^2$ of just 7 %.

That is, using this form of analysis, near New York City the early AIDS epidemic was coherently focused on the commuting center, with the distributions of different years actually obtained from those earlier by simple vertical displacement of the graph, according to multivariate analysis of covariance.

For the San Francisco metro region, however, each county had its own epidemic, and, in spite of the elevated rate for San Francisco itself, no regional coherence, in the sense defined above, was observed.

New York City and San Francisco have significantly different patterns of reaction against the Civil Rights Movement of the 1950's and 60's, i.e. of the revitalization of the US system of Apartheid. Wallace and Wallace (1998) describe in great detail how, after 1970, as the Civil Rights Movement turned its attention from its successes in the South to the ghettoized minority urban communities of the North, New York City embarked on a successful program of ethnic cleansing which came to be termed planned shrinkage, and was aimed at dispersing and disempowering ethnic minority voting blocks. Carried out in considerable part through the denial of essential municipal services to neighborhoods on the basis of race, this program resulted in a massive, uncontrollable outbreak of contagious urban decay – fires, building abandonment and forced population displacement – which left vast sectors of the city looking like Dresden after World War II.

San Francisco, on the other hand, applied a rapier rather than a meat ax in its dispersal of ethnic minority populations. A gentrification of the Black section of the city was carried out in the context of the expansion of nearby middle class neighborhoods, primarily the Gay ghetto of the Castro District: poor minorities were forced out economically, by relentlessly rising rents, and driven across San Francisco Bay into Oakland and Richmond, rather than displaced by a scorched earth policy as was the case in New York City.

New York City's uncontrolled outbreak of contagious urban desertification indeed dispersed minority voting blocks, causing a forced migration of people of color largely within the city, breaking the back of the neighborhood economies, the churches, local civic associations and other social structures particularly important for the socialization of the young. But the process also served to drive some 1.3 million non-Hispanic Whites – along with much of the Black middle class – from the city into the suburbs at a time when San Francisco's delicate touch made the central city more attractive to the middle class.

Apparently New York's version of the counterreformation against the Southern-based Civil Rights Movement tightened the ties binding metro regional counties – the commuting field – beyond a critical threshold, creating a unified disease ecosystem, while San Francisco's more sophisticated version of that counterreformation did not.

In both cases, however, extreme marginalization of Gay males, and their resulting concentration in small urban areas, did serve to create epicenters for AIDS. Different policies of racial apartheid, however, fundamentally altered subsequent metro regional disease spread.

Nationally and locally, then, in the US the early diffusion of AIDS was intimately associated with, if not driven by, the patterns and processes of the nation's system of Apartheid, as described, for example, by Massey and Denton (1993).

There is, it seems, far more to the diffusion of infectious disease than the dispersal of a dye marker along travel pathways, although that is clearly one part of the story. Here we will focus on the other part, uncovering a population-level epidemic quasi-language reflecting power relationships between groups within the underlying society.

**3. RECONSIDERING INFECTION** The approach is counterintuitive: One must, in essence, address generalized stochastic resonance in a spatial array of nonlinear components from an information theory perspective, a hard problem (R. Wallace, 2000).

The central idea of stochastic resonance is that the addition of noise to a weak input signal, usually taken as some repeated train of excitations, can raise the amplitude of the combined signal so as to exceed the trigger of a relatively powerful but highly nonlinear oscillator, resulting in an amplified train of output signals. Proper choice of the amplitude of the

noise can maximize the signal-to-noise ratio of the combined system. See, for example, Gammaitoni et al. (1998) for a review.

Explicit applications of a stochastic resonance viewpoint have been recently made to the triggering of epidemics by the noise of regressive social policies affecting the underlying signal of a marginalized community (e.g. Wallace et al. 1997; Wallace and Wallace, 1997a, b; Wallace et al., 1997; Wallace et al. 1999). Such treatments are inherently limited by the fact that long trains of output are not easily observed: Historically, multiple plague outbreaks, repeated economic catastrophes, episodes of mass slaughter and such like are self-limiting in the most Darwinian manner.

Each case history required a different elaborate mathematical attack. Wallace and Wallace (1997a), for example, studied the ongoing tuberculosis crisis in New York City from this viewpoint. Their analysis required the coupling of a model of contagious urban decay triggered by 'planned shrinkage' cuts in fire service provided racial minority voting blocks to a TB model, both having numerous simultaneous nonlinear differential equations.

Wallace (1999) used the Martingale Theorem to greatly simplify examination of coupled, spatially nested epidemics. Rather than seeking detailed time dynamics, such a treatment asks whether overall disease patterns are approaching higher or lower limiting states of endemicity or recurrence. Here we will ask similarly generic questions about stochastic resonators and the distribution of fluctuational paths leading to the triggering of a resonance, and, in fact, generalize the nested Martingale example accordingly.

McClintock and Luchinsky (1999) suggest a unifying perspective, looking at the prehistory probability density of the ensemble of pathways by which a system susceptible to stochastic resonance approaches the trigger. This leads directly to a formal development in the spirit of the Onsager and Machlup (1953) study of the distribution of fluctuational paths. The analysis, however, is carried out in terms of the Shannon-McMillan Theorem, one of the two basic foundations of information theory, and itself an asymptotic relation of probability as fundamental as the Martingale Theorem.

Others have, indeed, used a maximization of noise-dependent mutual information between input and amplifier output to characterize stochastic resonance (e.g. Deco and Schurmann, 1998; Henegan et al., 1996; Godivier and Chapeau-Blondeau, 1998; Schimansky-Geier et al., 1996). Here we attempt a more extensive analysis using these methods.

The modern mathematical context for such extension is a branch of applied probability theory known as large deviation theory (e.g. Dembo and Zeitouni, 1998; Ellis, 1985) which seeks to unite analysis of fluctuations, statistical mechanics and information theory under a single formalism. The first real step in this direction was taken by Cramer (1938). Subsequent proofs of the ergodic theorem, in the context of generalizations of Cramer's results by Gartner and Ellis, permit derivation of the Shannon-McMillan Theorem as the 'zero error limit' under the rubric of rate distortion theory (Dembo and Zeitouni, 1998).

Next, two mathematical infectious disease models are restated as stochastic resonances.

**4. THE SIMPLE EPIDEMIC** We first take a simple epidemic outbreak, with removal, to represent a large fluctuation in an otherwise relatively normal public health system. To paraphrase the paper by Dykman et al., (1996), large fluctuations, although infrequent, are fundamental in a broad range of processes, and it was recognized by Onsager and Machlup (1953) that insight into the problem could be gained from studying the distribution of fluctuational paths along which the system moves to a given state. This distribution is a fundamental characteristic of the fluctuation dynamics, and its understanding leads toward control of the fluctuations.

Again following Dyckman et al. (1996), fluctuational motion from the vicinity of a stable state may occur along different paths. For large fluctuations, the distribution of these paths peaks sharply at along an optimal, i.e. most probable, path. In the theory of large fluctuations, the pattern of optimal paths plays a role similar to that of the phase portrait in nonlinear dynamics.

In this context we restate the problem of the simple epidemic with removal as a stochastic resonance. More complicated models could be chosen, as in the next section, but similar results follow.

Let $X$ be the number of susceptible individuals in a population, $Y$ the number of infectives and $Z$ the number removed, by death or immunity. Let $t$ be the time. The simple epidemic has the form

$$
\begin{aligned}
dX/dt &= -\beta XY, \\
dY/dt &= \beta XY - \gamma Y \\
&= Y(\beta X - \gamma), \\
dZ/dt &= \gamma Y, \\
N &= X + Y + Z.
\end{aligned}
\tag{1}
$$

$\beta$ is the infectivity of the disease, and $\gamma$ the rate of removal of infective individuals, and $N$ is the total population.

Let $\rho \equiv \gamma/\beta$. Suppose at time $t = 0$ there are $X_0$ susceptible individuals. From the second of these expressions it becomes clear that no epidemic outbreak takes place if $X_0 < \rho$. If $X_0 = \rho + \delta, 0 < \delta \ll \rho$, then a simple calculation (e.g. Bailey, 1975) shows that the total number infected over the

course of the epidemic, and the final number of susceptibles after it, will be approximately

$$Z_\infty \approx 2\delta$$

$$X_\infty \approx \rho - \delta. \tag{2}$$

We suppose the epidemic process has a natural time frame relatively short with respect to periods of observation, $\Delta T$, so that epidemic outbreaks have time to equilibrate.

We look at the system at times $i = 0, 1, ...n$ normalized to multiples of $\Delta T$, with initial conditions $(X_0^i, \rho_i)$. These we write as points on a two dimensional graph labeled as $a_i$. We will call $a_i$ the 'state' of the system at time $i$.

The total number infected during an epidemic outbreak at time $i$ will be $Z_\infty^i \approx 2(X_0^i - \rho_i)$ if $X_0^i > \rho_i$, and 0 otherwise.

For marginalized communities – the epicenter of most disease outbreaks in developed countries – the state of the system at time $i$, $a_i = (X_0^i, \rho_i)$, is the result of the interaction between the socioeconomic opportunity structure available to it, i.e. the strictures of our Apartheid system, and the patterns of compensatory behavior developed by the community to address the impacts of those strictures. Coping strategies in marginalized communities are often characterized as risk behaviors by researchers based in the marginalizing superstructure, regardless of their inevitability under the pressure of constraint. There is much research on the topic, some from an information theory viewpoint (e.g. Wallace, Fullilove and Flisher, 1996; Wallace, Flisher and Fullilove, 1997; Wallace and Fullilove, 1999).

Figure 3 illustrates a revision of the simple epidemic with removal from this viewpoint. The community structure and the externally-defined opportunity structure in which it is embedded are convoluted together at times $i = 0, 1, ...n$ to create the *correlated* sequence of states $a_0, a_1, ...a_n$, which we will call a path $x$. That path enters the nonlinear amplifier of the epidemic process, producing an output sequence of epidemics of final sizes $Z_0, Z_1, ...Z_n$.

Figure 4 shows several such paths $x = a_0, a_1, ...a_n$ in the two-dimensional parameter space defined by the points $(X_0^i, \rho_i)$. Those for which $X_0^i < \rho_i$ do not lead to epidemic outbreaks, the region below the line $X = \rho$. We shall be particularly concerned with paths starting from an initial state $a_0$ below the line, and leading to some state $a_n$ above it.

That is, here we primarily focus on the properties of the paths which lead to large fluctuations in $Z$ rather than on the technical details of the nonlinear oscillator. Most current research in mathematical epidemiology appears limited to study of that oscillator, seemingly independent of larger, and indeed determining, contexts by design. See Wallace and Wallace (1997a) for a more detailed criticism.

To reemphasize the change in perspective, we note that the states constituting the paths of interest do not, in general, undergo simple uncorrelated Brownian motion: these paths have internal structure, serial correlations which contain essential information on systematic changes in community, constraints and their interactions. Thus Monte Carlo sensitivity analysis which randomly varies the parameters of some large array of simultaneous nonlinear differential equations modeling an epidemic is rather beside the point.

We have rephrased the simple epidemic as a classic stochastic resonance: The community structure is the signal, the opportunity structure the noise, and their convolution produces paths which then trigger a nonlinear amplifier whose output is the outbreak of infection.

**5. ENDEMIC INFECTION** A more sophisticated approach involves, again counterintuitively, asking a simpler question; does an established endemic infection remain the same, grow larger, or decline in time, rather than examining infection rates or processes in detail. The central tool for this is the Martingale Theorem, another of the fundamental asymptotic limit theorems of probability.

Suppose a player begins a game of chance with an initial fortune of some given amount, and bets $n$ times $(n = 1, 2, ...)$ according to a stochastic process in which a stochastic variable $\mathbf{X}_n$, which represents the size of the player's fortune at play $n$, takes values $\mathbf{X}_n = x_{n,i}$ with probabilities $P_{n,i}$ such that

$$\sum_i P_{n,i} = 1,$$

where $i$ represents a particular outcome at step $n$.

Assume for all $n$ there exists a value $0 < C < \infty$ such that the expectation of $\mathbf{X}_n$,

$$E(\mathbf{X}_n) \equiv \sum_i x_{n,i} P_{n,i} < C \tag{3}$$

for all $n$. That is, no infinite or endlessly increasing fortunes are permitted.

We note that the state $\mathbf{X}_n = 0$, having probability $P_n^0$, i.e. the loss of all a player's funds, terminates the game. Regarding HIV, the particular 'game' to which we will eventually apply this theory, for the foreseeable future,

$$P_n^0 \to 0.$$

That is, once an infection with a long latent period becomes established, its probability of extinction is essentially zero on the timescale of real events defined by the sequence $n$. We will assume the games played in our casino have this property. Indeed, the only epidemic game to terminate in this century is smallpox, which had a two hundred year prior history of safe, cheap, universally effective vaccination.

We suppose it possible to define conditional probabilities at step $n+1$ which depend on the way in which the value of $\mathbf{X}_n$ was reached, so that we can define the conditional expectation of $\mathbf{X}_{n+1}$:

$$E(\mathbf{X}_{n+1}|\mathbf{X}_1, \mathbf{X}_2, ...\mathbf{X}_n) \equiv E(\mathbf{X}_{n+1}|n)$$

The 'sample space' for the probabilities defining this conditional expectation is the set of different possible sequences of the $x_{m,i} > 0$:

$$x_{1,i}, x_{2,j}, x_{3,k}...x_{n,q}$$

We call the sequence of stochastic variables $\mathbf{X}_n$ defining the game:
a *Submartingale* if, at each step $n$,

$$E(\mathbf{X}_{n+1}|n) \geq \mathbf{X}_n,$$

a *Martingale* if

$$E(\mathbf{X}_{n+1}|n) = \mathbf{X}_n$$

and
a *Supermartingale* if

$$E(\mathbf{X}_{n+1}|n) \leq \mathbf{X}_n.$$

$\mathbf{X}_n$ is, remember, the player's fortune at step $n$.

Clearly a submartingale is favorable to the player, a Martingale is an absolutely fair game, and a supermartingale is favorable to the house.

Regardless of the complexity of the game, the details of the playing instruments, the ways of determining gains or loss or their amounts, or any other structural factors of the underlying stochastic process, the essential content of the Martingale Limit Theorem is that in all three cases the sequence of stochastic variables $\mathbf{X}_n$ converges in probability 'almost everywhere' to a well-defined stochastic variable $\mathbf{X}$ as $n \to \infty$. That is, for each kind of Martingale, no matter the actual sequence of winnings $x_{1,i}, x_{2,j}, ...x_{n,k}, x_{n+1,m}, ...$, you get to the same limiting stochastic variable $\mathbf{X}$. Sequences for which this does not happen have zero probability.

A simple proof of this result (e.g. Petersen, 1995) runs to several pages of dense mathematics using modern theories of abstract integration on sets.

To put these matters in perspective, we claim that the socioeconomic system is the house, the player is an established infectious agent like HIV, and the player's fortune at step $n$, $\mathbf{X}_n$, is the number of people infected. Each 'step' $n$ is determined by the recurrent patterns which structure our lives; the daily journey-to-work, the weekly ceremonies of worship or binge behavior, and the annual cycles of ritual and other gatherings, including those driven by the weather, which often determines the fraction of time spent indoors. Other inherent characteristic times may require aggregation of these 'natural' cycles.

A supermartingale is a declining infection, a submartingale is a spreading infection. Both, according to this theorem – one of the most powerful and subtle results of 20th century mathematics – will inevitably attain a limiting state of endemicity. This may fluctuate considerably and in a structured or serially correlated manner, but will do so about a mean value, $E(\mathbf{X})$, regardless of the complexity of the rules of the game.

Industrialized social systems are, of course, not simple analogs to gambling casinos, but reflect the large-scale patterns of nested hierarchy defined by their underlying economic engines (Abler, Adams and Gould, 1971).

We therefore propose a 'nested Martingale' model for epidemic spread in a hierarchically-structured social system. This will be a compound stochastic process in which the winnings at the smaller scale, played by one set of rules, contribute, in some sense, to a quite different game having completely different rules on a larger scale. These games are bounded, as we have discussed, by the conditions $E(\mathbf{X}_n) < C$, for some finite positive $C$, and $P_n^0 \to 0$.

The essential point is that a proportion of the winnings from the smaller game are duplicated by a benefactor and directly raise the magnitude of the player's fortune for the larger, embedding game.

If the inner game is characterized at step $n$ by the random variable $\mathbf{Y}_n$, then the real winnings at step $n+1$ for the embedding game, associated with the random variable $\mathbf{X}_{n+1}$, become, for some function $f_n$, which may involve additional stochastic variables,

$$\mathbf{X}_{n+1} = f_n(\mathbf{X}_n, \mathbf{Y}_n, \mathbf{Y}_{n+1}).$$

(4)

A slightly different approach would involve conditional expectations in the convolution of scales:

$$E(\mathbf{X}_{n+1}|n) = F_n(\mathbf{X}_n, \mathbf{Y}_n, E(\mathbf{Y}_{n+1}|n))$$

(5)

for some function $F_n$.

Traditionally the simplest version of this extension assumes that the compound game is simply a subset of the original. In our context this would be to envision, for example, a suburban county connected to an inner city by the commuting field (Wallace, et al., 1997) as functioning according to its own self-conception; an entity separated by social and geographic distance from the troubles of the deteriorating inner city, and having little of the kind of internal structure which would spread disease within its own borders. In this case the diseases of the suburb are entirely those of the larger city center, attenuated by segregation:

$$\mathbf{X}_{n+1} = \mathbf{X}_n + \mathbf{A}_n(\mathbf{Y}_{n+1} - \mathbf{Y}_n). \tag{6}$$

We assume $\mathbf{A}_n \geq 0$ is a non-negative stochastic variable, which can indeed take the value 0. The model states that the number of outlying cases at time $n+1$ will be the number at time $n$, augmented or decremented by the change in case numbers within the city center, as attenuated by the segregation filter $\mathbf{A}_n$. This may, for example, be greater than zero only one time in ten or a hundred, on average. Taking the conditional expectation gives

$$E(\mathbf{X}_{n+1}|n) = \mathbf{X}_n + \mathbf{A}_n(E(\mathbf{Y}_{n+1}|n) - \mathbf{Y}_n) \tag{7}$$

where we recognize the conditional expectation of any variate $\mathbf{Z}_n$ at step $n$ is just its value.

Since $\mathbf{A}_n \geq 0$, *the game described by the attenuated sequence* $\mathbf{X}_n$ *has the same martingale classification as does the nested central city game described by* $\mathbf{Y}_n$.

The $X$-processes in equation (6) is the *Martingale transform* of $\mathbf{Y}_n$ (Taylor, 1996, p.232; Billingsley, 1968, p. 412), and the result is classic, representing the *impossibility of a successful betting system.* That is, the betting system of moving to the suburbs to get away from urban problems, in the long run, can't win if the city center is itself losing.

Note that the basic Martingale transform can be rewritten as

$$\frac{\mathbf{X}_{n+1} - \mathbf{X}_n}{\mathbf{Y}_{n+1} - \mathbf{Y}_n} \equiv \frac{\Delta \mathbf{X}_n}{\Delta \mathbf{Y}_n} = \mathbf{A}_n,$$

or

$$\Delta \mathbf{X}_n = \mathbf{A}_n \Delta \mathbf{Y}_n. \tag{8}$$

Induction gives

$$\mathbf{X}_{n+1} = \mathbf{X}_0 + \sum_{j=1}^{n} \mathbf{A}_j \Delta \mathbf{Y}_j. \tag{9}$$

This notation is suggestive: in fact the Martingale transform is the discrete analog of Ito's stochastic integral relative to a sequence of stopping times, (Taylor, 1996, p. 232; Protter, 1990, p. 44; Ikeda and Watanabe, 1989, p. 48). In the stochastic integral context the $Y$-process is called the integrator and the $A$-process the integrand. Further development leads toward generalizations of Brownian motion, the Poisson process, and so on (Meyer, 1989; Protter, 1990).

The basic picture is, thus, of another stochastic resonance: the transmission of a signal, $\mathbf{Y}_n$, convoluted with a noise, $\mathbf{A}_n$. This 'noise' is, however, defined in our context by powerful factors of socioeconomic structure. As described above, however, detailed examination of the spread of AIDS and other pathologies in a number of metropolitan regions shows this is not always simple (Wallace et al., 1997).

A more realistic extension of the elementary denumerable Martingale transform for our purposes is

$$\mathbf{X}_{n+1} = \mathbf{X}_n + (\mathbf{B}_{n+1} - \mathbf{B}_n)\mathbf{X}_n + \mathbf{A}_n(\mathbf{Y}_{n+1} - \mathbf{Y}_n), \tag{10}$$

where $\mathbf{B}_n$ is a stochastic variable representing, for example, the balance between the growth and removal of infection, for example within a suburban county, while $\mathbf{A}_n$ again measures, for example, the coupling of the suburb to the central city. A positive difference, $\Delta \mathbf{B}_n = \mathbf{B}_{n+1} - \mathbf{B}_n > 0$, represents the local spread of infection.

Using the more suggestive notation of equations (8) and (9) this becomes the fundamental stochastic differential equation

$$\Delta \mathbf{X}_n = \mathbf{X}_n \Delta \mathbf{B}_n + \mathbf{A}_n \Delta \mathbf{Y}_n. \tag{11}$$

Taking conditional expectations gives

$$E(\mathbf{X}_{n+1}|n) - \mathbf{X}_n =$$

$$\mathbf{X}_n(E(\mathbf{B}_{n+1}|n) - \mathbf{B}_n) + \mathbf{A}_n(E(\mathbf{Y}_{n+1}|n) - \mathbf{Y}_n). \tag{12}$$

We can assume $\mathbf{X}_n, \mathbf{A}_n \geq 0$. The Martingale classification of suburban infection $\mathbf{X}$ thus depends on those of $\mathbf{B}$ and $\mathbf{Y}$. If the suburb has a stagnant public health system, so that $\mathbf{B}$ is a Martingale, then a growing infection within the city center – $\mathbf{Y}$ a submartingale – will express itself as a growing submartingale of disease within the suburb.

In general, however, the social deterioration within the city can be expected to express itself as neglect of the poor within nearby suburbs, if only from an outward diffusion of social disintegration which overwhelms local social and public health programs. That is, $\mathbf{B}$ is itself likely to become a submartingale, resulting in a very rapid spread of suburban disease.

Even if the suburb has a highly effective public health system, so that $\mathbf{B}$ is a supermartingale, the terms $\mathbf{A}_n \Delta \mathbf{Y}_n > 0$ will cause an increase in the endemic level of infection within the suburb, above what it had been.

If the terms $\mathbf{A}_n \Delta \mathbf{Y}_n > 0$ are sufficiently large, however, infection within the city center may overwhelm even the best suburban public health programs, turning local disease supermartingales into submartingales strongly linked to the central epidemic outbreak. The 'paths' for this process as a stochastic resonance which are analogous to those shown in figure 4 are sequences $x = (a_0, a_1, ..., a_n)$ with $a_j = (A_j \Delta Y_j, B_j)$.

**6. THE DUAL INFORMATION SOURCE** Next we formalize and extend the examples above, considering noise and signal of a generalized stochastic resonance first as mixed together according to some algorithm and then, given that mixing, examining the influence of a fixed signal form and a variable noise. It may be necessary to coarse grain the model, in much the same sense that symbolic dynamics discretizes a dynamical system (e.g. Beck and Schlogl, 1993).

Suppose a system has discrete (if necessary, coarse-grained) states $a_i$, where $i$ is a non-negative integer (for example, a given year), and that certain sequential patterns of these states – noise and signal together – of the form $a_0, a_1, ...a_n$, which we call paths $x$, lead to discontinuous observable events, an extremely general stochastic resonance analogous to the enhanced clicking of a switch – the nonlinear oscillator – by a weak signal in the presence of noise. That is, assume each path $x$ has associated with it a discontinuous function $h(x)$ taking possible values $0, 1$. Given an initial state $a_0$ such that $h(a_0) = 0$ we examine all possible subsequent paths $x$ beginning with $a_0$ and leading exactly once to the event $h(x) = 1$. Thus $h(a_0, ..a_j) = 0$ for all $j < m$ but $h(a_0, ...a_m) = 1$. We call such paths *meaningful*.

The definitions can be extended, under proper conditions, to a stochastic system in which $h(x)$ is the probability a nonlinear oscillator fires, provided a disjunction can be made between a relatively small number of paths $x$ which have a high probability of triggering the oscillator, and a large number with low probability which do not.

For the nested Martingale endemic infection, we are concerned with the condition that a declining infection, a supermargingale, turns into a rising infection, a submargingale. Thus $x = (a_0, ..., a_n)$ with $a_j \equiv (A_j \Delta Y_j, B_j)$, and $h(x) = 0$ if the system remains a supermartingale, while $h(x) = 1$ if it transforms into a submartingale.

For each positive integer $n$ let $N(n)$ be the number of meaningful, i.e. high probability, 'grammatical' and 'syntactical' paths of length $n$. We assume $N(n)$ to be considerably less than the number of all possible paths of length $n$ – stochastic resonance transitions are comparatively rare – and in particular assume that the finite limit

$$H = \lim_{n \to \infty} \frac{\log[N(n)]}{n} \tag{13}$$

exists and is independent of the path $x$. We shall call a stochastic resonance satisfying this condition *ergodic*.

Although we will not pursue the matter here, the underlying space of the $a_i$ can be partitioned into disjoint equivalence classes according to whether individual states can be connected by meaningful paths. This leads to a groupoid structure whose symmetry can be broken by internal crosstalk and is constrained by slower acting external structures. We will, however, examine the simplified dynamics of cross talk linkages below.

We can define an ergodic information source $\mathbf{X}$ associated with stochastic variates $X_i$ by having them take the values $a_i$ at time $i$ with joint and conditional probabilities $P[a_0, ..., a_i]$ and $P[a_i | a_0, a_1, ...a_{i-1}]$.

The joint uncertainty of two stochastic variates $X$ and $Y$, taking possible values $x_i$ and $y_i$, is defined in terms of their joint probabilities as

$$H(X, Y) = -\sum_i \sum_j P(x_i, y_j) \log[P(x_i, y_j)]. \tag{14}$$

The conditional uncertainty of $X$ given $Y$ is

$$H(X|Y) = -\sum_i \sum_j P(x_i, y_j) \log[P(y_j | x_i)]. \tag{15}$$

See Khinchin (1957, pp. 117-120) or Ash (1990) for more details.

By the Shannon-McMillan or Asymptotic Equipartition Theorem (AEPT), the source uncertainty of the ergodic information source $\mathbf{X}$ satisfies the relations

$$H[\mathbf{X}] = \lim_{n\to\infty} \frac{\log[N(n)]}{n}$$

$$= \lim_{n\to\infty} H(X_n|X_0...X_{n-1})$$

$$= \lim_{n\to\infty} \frac{H(X_0,...X_n)}{n+1}$$

(16)

For large $n$ the probability of a meaningful path of length $n$ is then $\propto \exp(-nH[\mathbf{X}])$.

We define the information source $\mathbf{X}$ as being *dual* to the generalized stochastic resonance. We have thus reduced three complex, interacting components into a single object on which we can impose important symmetries.

The utility of this will appear in due course.

Source uncertainty is a language function with an important heuristic interpretation (Ash, 1990, p. 206):

> "...[W]e may regard a portion of text in a particular language as being produced by an information source. The [conditional] probabilities $P[X_n = a_n|X_0 = a_0..., X_{n-1} = a_{n-1}]$ may be estimated from the available data about the language; in this way we can estimate the uncertainty associated with the language. A large uncertainty means... a large number of 'meaningful' sequences. Thus given two languages with uncertainties $H_1$ and $H_2$ respectively, if $H_1 > H_2$ then in the absence of noise it is easier to communicate in the first language; more can be said in the same amount of time. On the other hand, it will be easier to reconstruct a scrambled portion of text in the second language, since fewer of the possible sequences of length $n$ are meaningful."

Languages are most fundamentally characterized by strict patterns of internal relationship, for example grammar, syntax, and higher levels of organization. This development suggests that stochastic resonances representing infectious disease can be highly structured and may be studied and perhaps predicted by understanding the metalanguage in which they are embedded and indeed which they define.

According to this development, then, nonsense paths $x = a_0, ..., a_n$ which violate the grammar and syntax of a particular generalized stochastic resonance cannot trigger it.

We next must separate signal and noise, using the Shannon Coding Theorem, rather than the AEPT. See R. Wallace (2000) for a more complete treatment.

There may be, for a given signal and nonlinear oscillator, more than one noise capable of triggering a response. That is, different kinds of 'noise,' which may have variable internal structure and be colored in addition to having a simple amplitude, can be mixed in various ways – additive, multiplicative, integral convolution, etc. – with a given highly structured signal to trigger a particular nonlinear oscillator. Note that which is signal and which is noise remains ambiguous from this viewpoint.

Fixing signal and oscillator, we expect there to be a least upper bound $C$ – the capacity of the resonance as an information channel – such that for all possible convolutions of signal with noise into a trigger language $\mathbf{X}$ whose source uncertainty is $H[\mathbf{X}]$,

$$H[\mathbf{X}] \leq C.$$

(17)

Ash (1990), for example, discusses the origin and implications of this relation: one cannot send an error-free message faster than the capacity of the channel.

Fixing signal and oscillator, and following the arguments of Godivier and Chapeau-Blondeau (1998), the Shannon Coding Theorem ensures there will always be an optimal coding scheme involving the noise such that the rate of error-free throughput of the signal will reach any positive $R < C$. This suggests that if the noise is very simple, a white noise having a single amplitude parameter, for example, there will be an optimum value which maximizes total throughput, i.e. mutual information or signal to noise ratio.

Stochastic resonance recovered, and indeed generalized when noise is itself a complicated language, having grammar and syntax. In our particular context this result implies that, for any given community and infectious disease, there will be a pattern of constraints imposed by the external, embedding society which can maximize disease prevalence and incidence, turning a declining infection into a rising one.

**7. COHERENCE** Thus far we have restricted our attention to a single nonlinear oscillator and its properties when two input signals are mixed. Here we attempt to expand the development to systems of oscillators coupled socio-spatially, in the largest sense. We assume there are two kinds of coupling between individual simple oscillators, defining a two-stage hierarchy of organization. The first we will assume is reflexive, symmetric and transitive, permitting the division of the array into disjoint equivalence classes (Wallace and Wallace, 1998, 1999). We will call this a strong tie, in the tradition of sociology (Granovetter, 1973). The second coupling, which we characterize as weak, operates across all possible subdivisions of the array, and does not permit identification of disjoint equivalence classes. A physicist might characterize these as local and mean field forces, respectively.

We assume the index of strong ties remains constant, and permit the index of weak tie coupling, which we will call $T$, to vary and to characterize the array as a whole.

We also assume the array, signal, and noise depend on three parameters, two explicit and one implicit. The explicit are an external field strength analog $J$ which gives a direction to the phenomenon, and an inverse disorder parameter $K$ defined as $K = 1/T$, where $T$ is an index of the strength of the 'weak' ties which couple elements of the array without disjointly partitioning them.

We may, in the limit, set $J = 0$. Other explicit parameters can be added, of course, at the expense of complicating the analysis.

The implicit parameter, which we call $r$, is an inherent generalized length on which the phenomenon – including its direction and temperature-analog – is defined. That is, we can write $J$ and $K$ as functions of averages of the parameter $r$, which may be quite complex, having nothing to do with conventional ideas of space. For example Wallace and Wallace (1998) examine the role of social as well as of spatial separation within a population in determining the probability of weak ties between individuals or subgroups.

Rather than specify complicated patterns of individual dependence or interaction for signal, noise and elements of the array, we follow the direction of the previous sections and instead work entirely within the domain of the uncertainty of the ergodic information source dual to the large-scale stochastic resonance defined by the entirety of the coupled array, $H[K, J, \mathbf{X}]$. This draconian simplification enables us to directly obtain certain general results.

Taking only $K$ as significant for the moment, the fundamental relation

$$H(K) = \lim_{n\to\infty} \frac{\log[N(K)]}{n}$$

has the same form as the free energy density of a physical system. If $Z(K)$ is the partition function defined by the system's energy distribution, then the free energy density $F(K)$ is defined as

$$F(K) = \lim_{V\to\infty} \frac{\log[Z(K)]}{V}, \tag{18}$$

where $V$ is the system volume.

Generalizing in the spirit of the large deviations program of applied probability (e.g. Dembo and Zeitouni, 1998), imposition of invariance under a renormalization transform in the implicit parameter $r$ on the dual information source of the generalized ergodic stochastic resonance characterizing the array as a whole leads to expectation of both a critical point in $K$, $K_C$, reflecting a phase transition to or from collective behavior across the entire array, and of power laws for system behavior near $K_C$. See Wilson (1971) for calculational details, which are standard, and Wallace and Wallace (1998, 1999), Wallace et al. (2003) or Wallace (2005) for a more complete application to information sources.

Let $\kappa = (K_C - K)/K_C$ and take $\chi$ as the correlation length defining the average domain in $r$-space for which the dual information source is primarily characterized by strong ties. We begin averaging across $r$-space in terms of 'clumps' of length $R$, defining $J_R, K_R$ as $J, K$ for $R = 1$. Then, following the physical analog of Wilson (1971), we choose the renormalization symmetry relations as

$$H[K_R, J_R, \mathbf{X}] = R^D H[K, J, \mathbf{X}]$$

$$\chi(K_R, J_R) = \frac{\chi(K, J)}{R}, \tag{19}$$

where $D$ is a nonnegative real constant, possibly reflecting a fractal network structure.

Other symmetry relations – not necessarily based on simple physical analogs – may well be possible, perhaps permitting the grouping of stochastic resonances into disjoint equivalence classes (Wallace, 2005). For example discussion of the phase transition to instability in quasiperiodic orbits leads to renormalization relations of the form

$$f(x) = \alpha^2 f(\alpha^{-1} f(\alpha^{-1} x))$$

and

$$f(x) = \alpha f(\alpha f(\alpha^{-2} x)).$$

See McCauley (1993, p. 168) for details.

Then near $K_C$, for $J = 0$, some clever development and a simple series expansion of equation (9) (Wilson, 1971; Binney et al., 1995; Wallace and Wallace, 1998, 1999) gives

$$H = H_0 \kappa^{sD}$$

$$\chi = \chi_0 \kappa^{-s} \tag{20}$$

where $s$ is a positive real constant.

Again, $H$ is the dual source uncertainty of the entire array as a stochastic resonance and $\chi$ the average size the region dominated by 'strong' ties.

Further from the critical point matters are more complicated (Wilson, 1971).

We next attempt to estimate the size, in some sense, of the disjoint partition of the array into strongly interacting subsets.

Assume $K < K_C$ and that the rate of change of $\kappa$ remains constant as $K \rightarrow K_C$, so that $|d\kappa/dt| = 1/\tau_K$.

Further analogs with physical theory suggest there is a characteristic time constant for the phase transition, $\tau \equiv \tau_0/\kappa$, such that if changes in $\kappa$ take place on a timescale longer than $\tau$ for any given $\kappa$, we may expect the correlation length to remain in equilibrium with internal changes, resulting in very large fragment sizes in $r$-space.

Following Zurek (1985, 1996), we argue that the critical freezeout time $\hat{t}$, will occur at a system time $\hat{t} = \chi/|d\chi/dt|$ such that $\hat{t} = \tau$. Taking the derivative $d\chi/dt$, remembering that by definition $d\kappa/dt = 1/\tau_K$, gives

$$\frac{\chi}{|d\chi/dt|} = \frac{\kappa\tau_K}{s} = \frac{\tau_0}{\kappa}$$

so that

$$\kappa = \sqrt{s\tau_0/\tau_K}.$$

Substituting this value of $\kappa$ into the equation for correlation length, the expected size of fragments of the spatially distributed generalized stochastic resonance in $r$-space, $d(\hat{t})$, becomes

$$d \approx \chi_0(\frac{\tau_K}{s\tau_0})^{s/2}. \tag{21}$$

The more rapid the changes, the smaller $\tau_K$ and the smaller, and more numerous, on average, the resulting fragments.

Different renormalization symmetry relations than equation (19) would, of course, lead to different universal power laws.

It is clear that sudden critical point transition is possible in the opposite direction for this model. That is, the system can suddenly go from a set of independent, isolated and spatially fragmented strongly interacting resonators of some average diameter, $d$, firing more or less at random, into a single large, coherent, tightly interlinked and (socio)spatially extended system.

That is, disjoint, incoherent, strongly coupled subregions having independent and isolated epidemic outbreaks can become a single, massively coherent spatiotemporal structure – a spreading pandemic – if the probability of weak ties between the components increases beyond a threshold.

**8. INSTITUTIONAL COGNITION** Although we do not have the space to explore the details, these results are formally homologous to recent developments in cognitive theory. Wallace (2005a, b), for example, describes cognitive process explicitly in terms of a dual information source involving an algorithmic combination of internal and external signals leading to the choice of one out of a large set of possible responses. That analysis is likewise in terms of internally-correlated paths of mixed signal $x = (a_0, ..., a_n)$ and a nonlinear oscillator $h(x)$ whose outcome is the result of decision. This approach could be reexpressed using the generalized stochastic resonance treatment presented here. Indeed, neural networks are sometimes given a simplified stochastic resonance analysis, and block diagrams like figure 3 are often found in descriptions of neural phenomena.

Public policy and economic practice are, in part, outcomes of institutional cognition, i.e. of complex sequences of correlated choices made by dominant subgroups, in the context of embedding circumstances which creat a path dependence representing the imposition of contextual constraints determined in no small part by community history. Such choices have corollary outcomes in terms of infectious diseases, particularly those associated with socially-determined risk behaviors (Wallace et al., 1996): will there be an outbreak at all, will endemic infection rates rise or decline? One can take the Rate Distortion Manifold perspective of Wallace (2005a) to view disease outcomes as a simplified coding of more complicated processes of institutional cognition, i.e. patterns of resource allocation. The relation between infectious disease and marginalization has long been well understood, and disease structure can be viewed as a direct and clear expression of social process: Those in power make complex decisions which become coded as starkly simple patterns of illness and early death.

The development can be further expanded using network information theory to take formal account of embedding contexts. See Wallace (2005a) for calculational details. The effect is roughly similar to the role of context in consciousness theory. The essential point is that external contexts, which profoundly affect the course of institutional cognition, may change only slowly, while change in patterns of infectious disease expressing that cognition can be very rapid indeed. One must address a very complicated tripartite mutual information splitting criterion of the form

$$I(X|Y|Z)$$

where $X$ is the rapidly changing dual information source to the infectious disease stochastic resonance, $Y$ the less rapidly changing information source dual to social cognition, and $Z$ the (usually) slowly changing information source representing embedding context. Calculation may involve a complicated groupoid algebra.

**9. DISCUSSION AND CONCLUSIONS** Infectious diseases do not, in general, simply constitute a dye marker for urban sociogeography, although the overall pattern of social and geographic spread is certainly constrained by, and must be consistent with, the underlying sociogeography.

Consideration of the prehistory probability distribution and the distribution of fluctuational paths suggests the possibility of understanding the phenomenon in terms of a generalized stochastic resonance having a dual ergodic information source, a kind of language. Paths of signal – community structure – convoluted with noise – the embedding opportunity structure – which are not consistent with the grammar and syntax of

that language cannot trigger simple epidemic outbreaks, or, taking the nested Martingale perspective, turn a declining infection into a rising one.

Conversely, for a given fixed signal – community structure – there will be an optimal noise – opportunity structure – whose convolution, analogous to coding, permits the system to approach a channel capacity measure arbitrarily closely. That is, certain patterns of constricted opportunity will most efficiently trigger repeated outbreaks of infectious epidemic disease in marginalized communities, or turn a falling endemic infection into a growing one.

Further development shows that spatially (or socio-spatially) distributed systems undergoing such generalized stochastic resonance can be subject to phase transition at critical values of driving parameters, which may encompass signal and non-linear amplifier as well as noise. These transitions should follow 'universal' power laws, depending on the appropriate renormalization symmetry, which is itself related to the underlying sociogeographic architecture.

Conversely, this analysis suggests isolated, incoherent spatiotemporal infectious epidemic resonators – individual neighborhoods, counties, or metropolitan regions – may suddenly coalesce into a single powerful and coherent amplifier, a spatially contagious or hierarchically spreading pandemic, under the influence of particular constrained opportunity structures.

This result has formal similarities to recent developments in cognitive theory, and suggests that diseases of marginalization, and their control, are very much creatures of institutional cognition, related to overt decisions regarding allocation of resources.

The analysis by Wallace et al. (1997) on the regional conformations of the early stages of AIDS in eight US metropolitan areas seems to provide a case history. As discussed, the two principal US AIDS epicenters of San Francisco and New York City showed markedly different forms of regionalization. New York had a coherent disease pattern across the entire metropolitan area, strongly dominated by the commuting center of Manhattan, while the San Francisco metro area, at least for the period studied, remained a patchwork of several individual outbreaks.

As described earlier, the two cities took significantly different approaches to reinforcement of the system of US Apartheid following the challenges of the Southern-based Civil Rights Movement. San Francisco made the city center attractive to White middle class residents, while New York drove some 1.3 million non-Hispanic Whites and other middle class residents from the city to the suburbs, tightening the weak ties linking counties of the metro region beyond threshold, and creating a unified disease ecosystem.

The synergistic unification of community, policy, and epidemic infectious disease into a language of power relations defined by the generalized stochastic resonance of emerging infectious disease suggests that public health interventions against plague must be similarly synergistic and hierarchical to bring the system below coherent threshold.

Our analysis particularly suggests that marginalized subgroups constitute keystone populations for the ecology of infectious disease, particularly AIDS. The most marginalized groups in the largest cities become the most central: For the US, as go New York City's South Bronx and Los Angeles' South Central, so goes the nation.

From the viewpoint of this work, then, victim and victimizer within the highly patterned, formally Manichean structures of an Apartheid system (Fanon, 1966) or other highly exploitative state relation will become jointly enmeshed in pandemic infectious disease. Apartheid and pestilence are thus ever two faces of the same coin.

This generalized treatment washes out confusing model-dependent behaviors which have been the focus of previous study, leaving behind a starkly simple structure in which the concentration of disease resulting from policies of marginalization represents, not containment, but intensification of the Paradox of Apartheid, where the powerful are ever more closely bound to the fate of their victims.

Application of this perspective to the emerging plague of MDR-HIV seems fairly direct. The 'Treatment Culture' which has dominated both official and nongovernmental organizations' AIDS policy in the US since the development of HAART is ending as HIV inevitably evolves resistance to drug regimens. An alternative evolutionary strategy for the virus would be to develop greater virulence, i.e. higher infectivity and a shortened or absent asymptomatic period (R.G. Wallace, 2004; Simon et al., 2003). Vaccine strategies seem similarly challenged by HIV's protean evolutionary nature.

As the AIDS Treatment and Vaccine subcultures disintegrate under the relentless pressure of pathogen evolution – as the American God of the Technical Fix fails – new social organizations must emerge to confront the disease. Traditional public health approaches, which address underlying structural factors responsible for disease incubation and spread at the population level – primarily the power relations between groups – have largely been abandoned in the US for obvious political reasons. The field is now dominated by a kind of rightist intellectual analog to the Marxism-Leninism studies of the fallen Soviet Union, effectively a Center-Right Lysenkoism strongly driven by funding patterns.

Controlling MDR and vaccine-resistant HIV will require resurrection of traditional public health, but this will be difficult because so much of the discipline's history has been lost in favor of the blame-the-victim, medicalized, and individual-oriented perspectives now popular with the current crop of major AIDS funding agencies and their client organizations. Many resulting projects are characterized as 'fundable trivialities' or 'planting a tree in a desert' by even those providing financial support.

MDR-HIV is poised to spread from traditional HIV epicenters to the rest of the US in much the same manner the pre-HAART pandemic spread nationally. In contrast, evidence exists that, for at least one more egalitarian social system – Amsterdam – there is a declining trend in transmission of drug-resistant HIV (Bezemer et al., 2004). Absent, however, some form of regime change in the US, the nation's response to the Katrina disaster affecting New Orleans provides a sentinel case history for the likely outcome.

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## FIGURE CAPTIONS

**Figure 1.** Log number of AIDS cases in the 25 largest US metropolitan regions, through 4/91 and 4/91-6/95. The composite index is X = .764 Log(USVC91) + .827 Log(USME87/USME72) + .299 Log(Prob. NY). USVC91 is the number of violent crimes, USMEnm the number of manufacturing jobs in year nm, and (Prob. NY) the probability of contact with New York City according to Census migration data for 1985-1990. Applying multivariate analysis of covariance, the two lines are parallel with different intercepts: The second is obtained simply by raising the first. This suggests a coherent, national-scale, spatiotemporal stochastic resonance linking marginalized inner-city communities, the Apartheid system which marginalizes them, and the epidemic outbreak which began within them, to the rest of the country.

**Figure 2.** Parallel regression lines for 24 counties of the New York Metropolitan Region. Y-axis is the log of the cumulative AIDS cases per 100,000, plotted versus a composite index $X = .759 \log(P_P) + .197 \log(\Delta\mu/\Delta A)$. $P_P$ is the percent of a county's population living in poverty and $\Delta\mu/\Delta A$ is the area density of the equilibrium commuting intensity. Again, the later period is obtained from the earlier by simple vertical displacement.

**Figure 3.** Restatement of the simple epidemic with removal as a stochastic resonance. The community structure and opportunity structure are convoluted at time $i$ into a 'state' $a_i$ defined by the initial conditions $(X_0^i, \rho_i = \gamma_i/\beta_i)$. If $X_0^i > \rho_i$ then the nonlinear amplifier of the epidemic process produces an output $Z_i \approx 2(X_0^i - \rho_i)$. Otherwise $Z_i = 0$. The nested Martingale argument is somewhat more complex, with $a_n = (A_n \Delta Y_n, B_n)$.

**Figure 4.** 'Paths' in the state space defined by $X_0$ and $\rho$ of figure 3. These all start at $a_0$ and terminate when the line $X = \rho$ is crossed and an outbreak occurs. The states $a_0, a_1, ...a_n$ constituting a path are *not* undergoing uncorrelated Brownian motion, and indeed the internal correlation properties of paths are very much the focus of interest. Similarly, for the nested Martingale example, the (coarse-grained)

$x = (a_0, ..., a_n)$ are also subject to a complex grammar and syntax defined by the power relations between marginalized and dominant subgroups.

Fig. 1

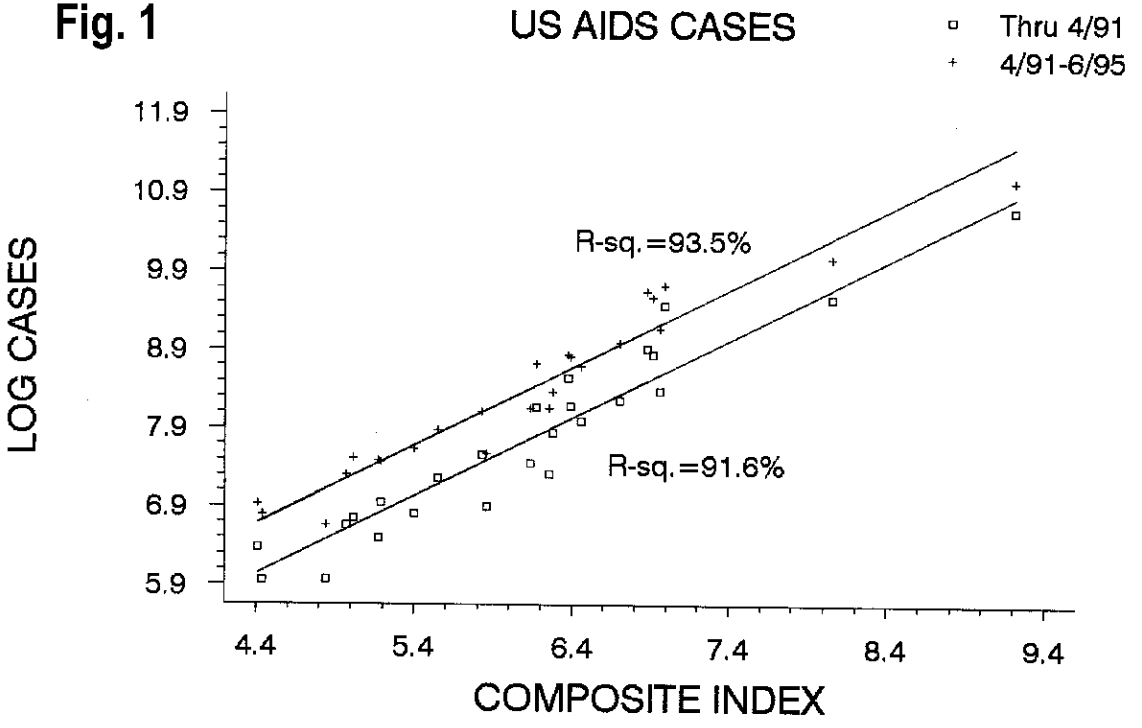

US AIDS CASES
Thru 4/91
4/91-6/95
LOG CASES
11.9
10.9
9.9
8.9
7.9
6.9
5.9
R-sq.=93.5%
R-sq.=91.6%
4.4
5.4
6.4
7.4
8.4
9.4
COMPOSITE INDEX

Fig. 2

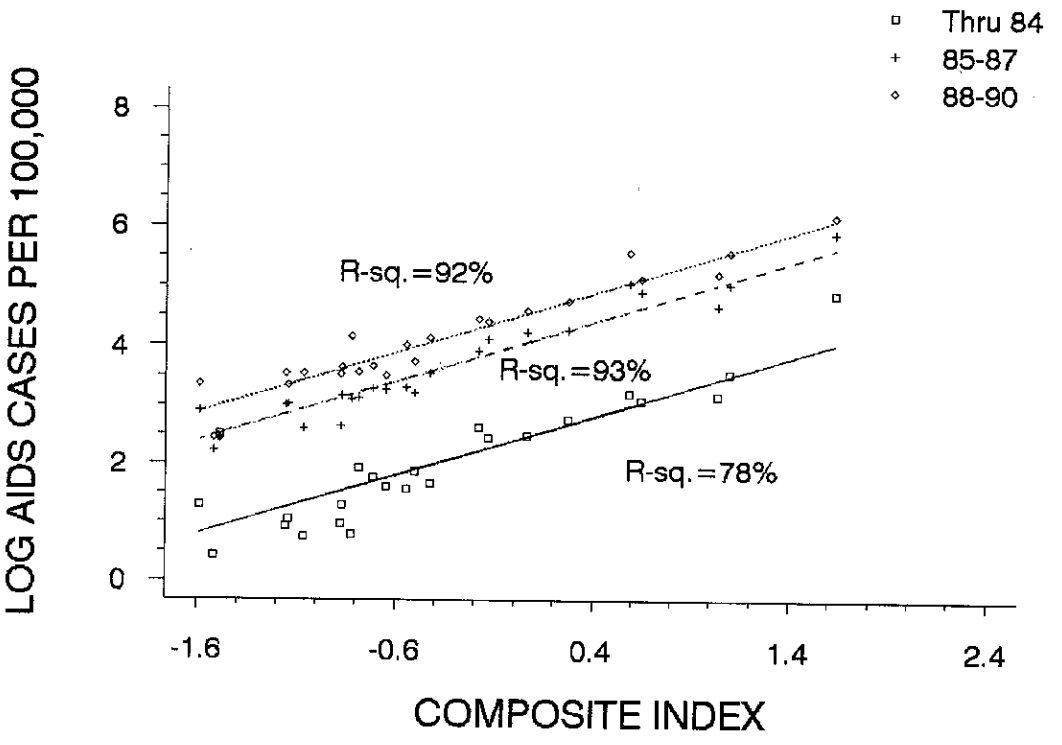
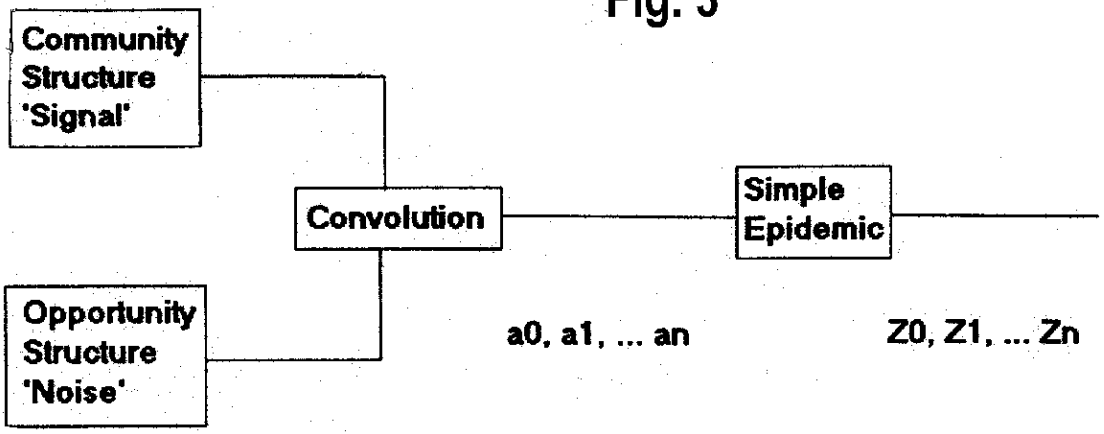

Thru 84
85-87
88-90
LOG AIDS CASES PER 100,000
8
6
4
2
0
R-sq.=92%
R-sq.=93%
R-sq.=78%
-1.6
-0.6
0.4
1.4
2.4
COMPOSITE INDEX

Fig. 3




PATH DIAGRAMS FOR THE SIMPLE EPIDEMIC

Fig. 4